ORIGINAL ARTICLE

Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China

T W Wong, W S Tam, T S Yu, A H S Wong

Occup Environ Med 2002;**59**:30–35

See end of article for authors' affiliations

Correspondence to: Professor T W Wong, Department of Community and Family Medicine, The Chinese University of Hong Kong, 4/F School of Public Health, Prince of Wales Hospital, Shatin, N T, Hong Kong; twwong@cuhk.edu.hk

Accepted 12 July 2001

Objective: To investigate the association between ambient concentrations of air pollutants and respiratory and cardiovascular mortalities in Hong Kong.
Methods: Retrospective ecological study. A Poisson regression of concentrations of daily air pollutants on daily mortalities for respiratory and cardiovascular diseases in Hong Kong from 1995 to the end of 1998 was performed using the air pollution and health: the European approach (APHEA) protocol. The effects of time trend, seasonal variations, temperature, and humidity were adjusted. Autocorrelation and overdispersion were corrected. Daily concentrations of nitrogen dioxide (NO_2), sulphur dioxide (SO_2), ozone (O_3), and particulate matter <10 μm in aerodynamic diameter (PM_{10}) were averaged from eight monitoring stations in Hong Kong. Relative risks (RRs) of respiratory and cardiovascular mortalities (per 10 μg/m^3 increase in air pollutant concentration) were calculated.
Results: Significant associations were found between mortalities for all respiratory diseases and ischaemic heart diseases (IHD) and the concentrations of all pollutants when analysed singly. The RRs for all respiratory mortalities (for a 10 μg/m^3 increase in the concentration of a pollutant) ranged from 1.008 (for PM_{10}) to 1.015 (for SO_2) and were higher for chronic obstructive pulmonary diseases (COPD) with all pollutants except SO_2, ranging from 1.017 (for PM_{10}) to 1.034 (for O_3). RRs for IHD ranged from 1.009 (for O_3) to 1.028 (for SO_2). In a multipollutant model, O_3 and SO_2 were significantly associated with all respiratory mortalities, whereas NO_2 was associated with mortality from IHD. No interactions were detected between any of the pollutants or with the winter season. A dose-response effect was evident for all air pollutants. Harvesting was not found in the short term.
Conclusions: Mortality risks were detected at current ambient concentrations of air pollutants. The associations with the particulates and some gaseous pollutants when analysed singly were consistent with many reported in temperate countries. PM_{10} was not associated with respiratory or cardiovascular mortalities in multipollutant analyses.

Many time series studies on the effects of variations in daily air pollutants on mortalities and morbidities have been reported in the United States and in Europe.[1–6] The role of particulates has been the focus of the research, and the associations have been found between total suspended particulates and particulates less than 10 μm in aerodynamic diameter (PM_{10}) and all mortalities and those caused by respiratory and cardiovascular diseases.[2 3 5–11] Associations have also been reported with gaseous air pollutants—namely, ozone (O_3),[4 12] nitrogen dioxide (NO_2),[4] sulphur dioxide (SO_2),[5 10 13 14] and carbon monoxide (CO).[14 15] Compared with the particulates, the relations between gaseous pollutants and mortalities are less consistent. Mortality has been shown to be associated with climate,[16] and its interaction with air pollutants have been reported in several European countries.[15 17] In Asia, few such studies have been conducted except in northern China (Beijing) and Korea.[7 18 19] The relations between seasons and air pollutants are likely to differ in tropical climates. Studies from tropical countries are scarce, possibly due to the lack of credible data. Hong Kong is a densely populated coastal city in southern China with 6.8 million people in a land area of about 1000 km^2. Summers are hot and humid, whereas winters are typically mild and dry. A large proportion of the population live in high rise buildings in close proximity to road traffic, a major source of air pollutants. Diesel vehicles contribute a substantial proportion of respirable particulates. Concentrations of PM_{10}, NO_2, and O_3 are higher than those in major cities in the United States, Western Europe, and some Asian countries such as Singapore and Japan, whereas SO_2 and CO concentrations are comparatively low.[20] Air pollutants are monitored systematically over most districts. The methods and quality of data are comparable with standards in many developed countries. A vigorous validation procedure of the air pollutant measurements is followed. Mortality statistics are comprehensive and systematically validated by the health authority. Owing to the relatively high concentrations of particulates and the oxidant pollutants (NO_2 and O_3) in Hong Kong and the proximity of the residences to the pollution source compared with western cities, an investigation into the association between the air pollutants and health outcomes is warranted. We have reported an association between daily hospital admissions and air pollutants.[21] To investigate the association between air pollution and mortality, and to compare the results with our previous findings and those reported elsewhere, we performed a time series analysis on concentrations of air pollutants and daily mortality data for respiratory and cardiovascular diseases over a 4 year period from 1995–8.

MATERIALS AND METHODS

Mortality data

Daily mortality data between 1995 and 1998 were obtained from the Census and Statistics Department. Mortality data covered all deaths reported in Hong Kong, and were coded

Abbreviations: APHEA, air pollution and health; the European approach; PM_{10}, particulate matter <10 μm in aerodynamic diameter; IHD, ischaemic heart diseases; COPD, chronic obstructive pulmonary diseases

according to the 9th revision of the international classification of diseases (ICD). Daily time series datasets were constructed for mortalities from "all diseases of the respiratory system" (ICD 461-519) and its subsets, chronic obstructive pulmonary diseases (COPD, ICD 490-496) and pneumonia and influenza (ICD 480-7). Daily datasets were also constructed for mortalities from "all diseases of the cardiovascular system" (ICD 390-459) and its subsets, ischaemic heart disease (IHD, ICD 410-414) and cerebrovascular disease (ICD 430-8).

Air quality and weather data

Air pollution data between 1995 and 1998 were obtained from the Environmental Protection Department. Hourly concentrations of four air pollutants: SO_2, NO_2, PM_{10}, and O_3 were monitored in eight monitoring stations interspersed in different districts of Hong Kong using pulsed fluorescence, gas phase chemiluminescence, tapered element oscillating microbalance, and ultraviolet absorption, respectively.[20] Twenty four hour mean concentrations of NO_2, PM_{10}, and SO_2 were calculated. As the formation of O_3 is dependent on sunlight, a daytime (0900–1700) 8 hour mean concentration of O_3 was used for analysis.[22] In view of the low ambient concentration of CO in Hong Kong, monitoring of CO has been confined to only one station in recent years. We have therefore excluded CO from our study. Daily mean temperatures and relative humidity for the same period were obtained from the Hong Kong Observatory.

Statistical analysis

A Poisson regression model was constructed in accordance with the air pollution and health: the European approach (APHEA) protocol.[22] The following terms were included to construct the core model: day of the time series (t), days of the week, trigonometric functions to control for seasonal variations ($\sin 2k\pi t/365$ and $\cos 2k\pi t/365$, where $k=1, 2, 3, 4$, and 6, represent cycles of 12, 6, 4, 3, and 2 months respectively), temperature and humidity.[21–23] To control for overdispersion, the covariance matrix was modified by multiplying the dispersion parameter ϕ, and the scaled deviance and log likelihoods used in likelihood ratio tests were divided by ϕ. The function obtained by dividing a log likelihood for the Poisson distribution by a dispersion parameter is an example of a quasi-likelihood function.[24 25] To control for autocorrelation, the autocorrelation functions plot of the residuals was examined and significant terms, up to lag day 7, were retained in the model.[23 26] Different pollutants may affect mortality with variable time lags. To test the influence of an individual pollutant on mortality, a single pollutant model was constructed for each of the four pollutants by adding its daily concentration to the core model. The best fitting lag period of each pollutant was found by testing its concentration on single lag days and on cumulative lag days (moving averages). Lag days from 0 (same day) to 5 (5 days before) were tested for O_3; lag days from 0 to 3 were tested for the other three pollutants. The lag period used to construct the model that gave the smallest Akaike's information criterion value was then selected for each pollutant.[27] The relative risk (RR) and its 95% confidence intervals (95% CIs) for a 10 $\mu g/m^3$ increase in the concentration of each pollutant were then calculated. To study the combined effects of the pollutants, multipollutant models were constructed. Firstly, all four pollutants (as continuous variables) were entered into the core model irrespective of significance. Non-significant ($p \geq 0.05$) terms were then successively removed from the model by a process of backward elimination, until only the significant terms remained. Owing to the non-trivial correlation of some pollutants, a pairwise approach was adopted as in the APHEA protocol if more than one pollutant seemed to be associated with the outcome, the associations with one pollutant stratified by the level of the other pollutant was sought.[22] With this approach, we explored the interaction between different pollutants by analysing each pair of pollutants using one as a continuous variable and the other as high and low concentrations dichotomised by the median, and their interaction term (between a pollutant and a high concentration of another pollutant). The influence of the concentration of a pollutant (expressed as a continuous variable) on a high and low concentration of another pollutant was then studied.[22 23] Interaction was considered to be present when the p value of the interaction term was less than 0.05. As an ambient temperature has a significant influence on mortality,[16] we also explored the possible interaction of cold seasons (December to February, when the mean monthly temperature was below 20°C) with the effects of the air pollutants by introducing a dummy variable for season in the model.[22] To investigate dose-response relations, we derived from the single pollutant models the respective RRs of daily deaths by pollutant concentrations categorised into deciles with the lowest decile as reference.[2]

Harvesting, a hypothesis that a high death toll on one day due to air pollution would deplete the pool of vulnerable people and result in fewer deaths on succeeding days, was investigated according to the procedure described by Spix *et al*.[28] In short, the coefficients of the interaction between the mean daily mortalities on the previous k days (where k ranged from 2 to 21) and each of the pollutants were checked. Harvesting was considered to be present if any of the coefficients was significantly negative.

RESULTS

During the 4 years, there were 128 229 deaths of which 58 347 (46%) were caused by respiratory and circulatory diseases. Table 1 shows the daily number of deaths by cause and the

Table 1 Summary statistics of daily numbers of deaths, concentrations of pollutants ($\mu g/m^3$), and weather data

	Mean	SD	Min	25%	Median	75%	Max
Causes of death (n):							
All respiratory diseases	17	5	3	13	17	20	33
Chronic obstructive pulmonary diseases	6	32	0	4	5	7	19
Pneumonia and influenza	10	4	1	8	10	13	24
All cardiovascular diseases	23	7	7	19	22	27	53
Ischaemic heart disease	9	3	1	6	9	11	29
Cerebrovascular disease	9	3	0	6	8	11	22
Air pollutants ($\mu g/m^3$):							
NO_2	56.40	19.24	15.29	43.29	54.19	68.10	151.5
PM_{10}	51.53	24.79	14.05	31.58	45.90	66.51	163.79
O_3	33.93	23.15	0.3	15.92	29.29	49.67	168.93
SO_2	16.68	11.59	1.05	8.58	13.97	21.27	90.06
Weather variables							
Temperature	23.4	5.0	6.9	19.2	24.6	27.6	31.3
Humidity	78.1	10.4	31	74	79	85	97

Table 2 Correlation between pollutants and weather variables

	SO_2	NO_2	PM_{10}	O_3	Temperature	Humidity
SO_2	1	0.438	0.344	–0.073	0.186	–0.089
NO_2		1	0.780	0.413	–0.346	–0.341
PM_{10}			1	0.538	–0.319	–0.524
O_3				1	–0.045	–0.530
Temperature					1	0.227
Humidity						1

pollutant concentrations. The Pearson's correlation coefficients between air pollutants and meteorological variables are presented in table 2. After fitting the core model, residuals were plotted against the predicted values for diagnostic checking. No cyclical pattern could be discerned in the residual plot. Table 3 summarises the RRs of daily deaths for different disease categories. The lag days for the pollutants that fitted the model best for all respiratory diseases varied for different pollutants, ranging from 1 to 2 single lag days and 0 to 1 cumulative lag days. For all cardiovascular diseases, the "best lag" ranged from 0 to 2 single lag days and up to 0 to 2 cumulative lag days. The overdispersion parameter (ϕ) was 1.14 and 1.09 respectively for respiratory and cardiovascular diseases. The autocorrelation coefficients (r) of the models ranged from 0.005 to 0.052. For all four pollutants, there was a significant increase in mortality for respiratory diseases that ranged from 0.8% to 1.5% per 10 $\mu g/m^3$ increase in concentration of pollutant. For COPD, the risks were higher and significant for all pollutants except SO_2. For pneumonia and influenza, the RRs were significant for NO_2 and SO_2 only. For all cardiovascular diseases and cerebrovascular diseases, the RRs for the best fitting lag days of all four pollutants were non-significant. A significant increase in mortality from IHD, ranging from 0.9% to 2.8%, was associated with a 10 $\mu g/m^3$ increase in the concentration of all four pollutants.

In the multipollutant model for all respiratory mortalities, SO_2 (RR=1.015) and O_3 (RR=1.010) remained after eliminating the non-significant pollutants (table 4). In the three and four pollutant models, O_3 was the only significant pollutant. The RR of O_3 was stable in all three models, and similar in magnitude to that in the single pollutant model. For COPD, O_3 was the only significant pollutant in the two, three and four pollutant models, with RRs slightly lower than in the single pollutant model. For pneumonia and influenza, SO_2 was significant after eliminating non-significant terms from the model. For IHD mortalities, NO_2 was the only significant pollutant in the four, three, and two pollutant models, its RRs being smaller than that in the single pollutant model.

No significant interaction between any pair of pollutants was found in the pairwise analyses, or between any pollutant and the cold season. None of the coefficients of the interaction terms between the pollution concentration and the mean mortalities of the previous days from lag day 2 up to day 21 were significantly negative, suggesting the absence of a harvesting effect. When RRs of deciles of the pollutant concentrations were derived from each single pollutant model, with the lowest decile as reference, an upward trend in RRs was found for all pollutants from the lowest to the highest deciles (fig 1).

Table 3 RRs (95% CIs)/10 μg/m^3 increase in concentration of pollutant for daily numbers of deaths from different diseases by pollutants

	SO_2	O_3	NO_2	PM_{10}
Respiratory diseases	Lag 0–1 days	Lag 2 days	Lag 0–1 days	Lag 1 day
RR	1.015	1.010	1.013	1.008
95% CI	(1.001 to 1.029)	(1.004 to 1.016)	(1.004 to 1.022)	(1.001 to 1.014)
Chronic obstructive pulmonary diseases	Lag 2 days	Lag 0–4 days	Lag 0–2 days	Lag 0–3 days
RR	1.010	1.034	1.023	1.017
95% CI	(0.990 to 1.029)	(1.017 to 1.052)	(1.006 to 1.041)	(1.002 to 1.033)
Pneumonia and influenza	Lag 0–1 days	Lag 2 days	Lag 0–3 days	Lag 2 days
RR	1.021	1.007	1.016	1.007
95% CI	(1.003 to 1.039)	(0.999 to 1.015)	(1.002 to 1.030)	(0.999 to 1.015)
Cardiovascular diseases	Lag 0–1 days	Lag 0 day	Lag 0–2 days	Lag 2 days
RR	1.007	0.997	1.008	1.003
95% CI	(0.994 to 1.020)	(0.991 to 1.003)	(0.999 to 1.016)	(0.998 to 1.008)
Ischaemic heart disease	Lag 1 day	Lag 3 days	Lag 1 day	Lag 0–3 days
RR	1.028	1.009	1.024	1.013
95% CI	(1.012 to 1.044)	(1.000 to 1.018)	(1.012 to 1.036)	(1.001 to 1.025)
Cerebrovascular disease	Lag 2 days	Lag 0 day	Lag 1 day	Lag 2 days
RR	0.9881	0.999	0.996	1.007
95% CI	(0.965 to 1.011)	(0.998 to 1.000)	(0.985 to 1.007)	(0.998 to 1.016)

Table 4 RRs (95% CIs) for daily numbers of deaths/10 μg/m³ increase in concentration of pollutant in multipollutant models*

Model	SO_2	O_3	NO_2	PM_{10}
All respiratory mortalities:				
4 Pollutant model	1.009 (0.990 to 1.029)	1.009 (1.002 to 1.016)	1.001 (0.991 to 1.020)	1.005 (0.992 to 1.010)
3 Pollutant model	1.010 (0.990 to 1.030)	1.009 (1.002 to 1.016)	1.006 (0.993 to 1.019)	
2 Pollutant model	1.015 (1.001 to 1.031)	1.010 (1.003 to 1.017)		
Mortality from COPD:				
4 Pollutant model	1.007 (0.9848 to 1.0296)	1.033 (1.0126 to 1.0548)	1.014 (0.989 to 1.041)	0.991 (0.968 to 1.015)
3 Pollutant model		1.032 (1.012 to 1.053)	1.016 (0.991 to 1.042)	0.993 (0.970 to 1.016)
2 Pollutant model*		1.029 (1.011 to 1.049)	1.011 (0.992 to 1.031)	
Mortality from pneumonia and influenza:				
4 Pollutant model	1.018 (0.997 to 1.040)	1.006 (0.997 to 1.015)	1.004 (1.017 to 1.025)	1.002 (0.991 to 1.013)
3 Pollutant model	1.017 (0.996 to 1.039)	1.006 (0.997 to 1.016)	1.006 (0.989 to 1.024)	
2 Pollutant model	1.021 (1.003 to 1.039)	1.008 (0.999 to 1.016)		
Mortality from ischaemic heart diseases:				
4 Pollutant model	1.011 (0.990 to 1.033)	1.007 (0.998 to 1.017)	1.020 (1.003 to 1.035)	0.9940 (0.978 to 1.009)
3 Pollutant model	1.012 (0.991 to 1.033)	1.006 (0.998 to 1.015)	1.016 (1.002 to 1.032)	
2 Pollutant model		1.006 (0.997 to 1.014)	1.022 (1.011 to 1.003)	

*Backward elimination of non-significant terms (p>0.05) starting with four pollutants.

DISCUSSION

This study provides additional information for our previous study on hospital admissions,[21] and the many time series studies on air pollution and mortality in temperate countries.[1–11 13 15 17–19 28 29 33–35 38 39] Although hospital admissions are influenced by socioeconomic and personal factors, mortality is the ultimate and most serious of all health outcomes. As explained earlier, both the mortality and air pollutant datasets were of reasonable quality. Our statistical methods followed the APHEA protocol, which facilitated the comparison of results. Significant associations were found with respiratory mortalities for all four pollutants in the single pollutant models. For the different respiratory diseases, the best lags ranged from 1 to 2 single lag days and 0 to 4 cumulative lag days. These lag periods were statistically chosen by model fitness. The durations of the lag periods, however, seemed reasonable, if we assume a short interval (in terms of several days) between exposure and death, which varies with different diseases. It is also reasonable to assume that mortality might be associated with cumulative exposure over several days. The risk estimates for respiratory mortalities, at 0.8% to 1.5% per 10μg/m³ increase in pollutant concentration were similar in magnitude to results reported elsewhere.[30] These mortality risks were, however, lower than risks of hospital admissions we reported previously (1.3% to 2.2% per 10 μg/m³ increase).[21] Of the respiratory diseases, the RRs for mortalities from COPD, the most sensitive mortality to air pollution, were higher for O_3 and NO_2 (at increases of 3.4% and 2.3% respectively). The RRs of cardiovascular mortalities were non-significant for all four pollutants, by contrast with our previous findings for hospital admissions for cardiovascular diseases.[21] Mortalities for IHD were significantly associated with all four pollutants, suggesting that the associations of air pollution were disease specific. Of cardiovascular diseases, the RRs of deaths from IHD (at 1.028 and 1.024 respectively) were

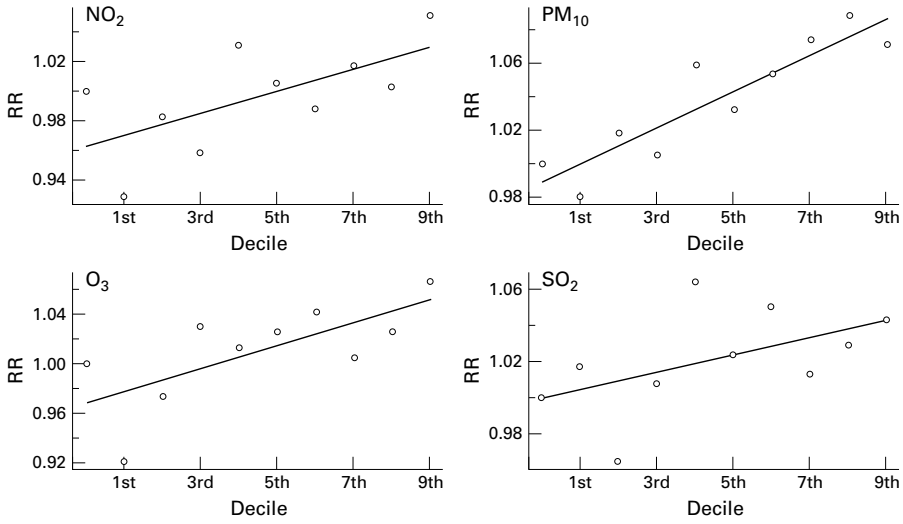

Figure 1 A regression line is fitted to show the upward trend of the RRs. RRs were derived from the Poisson regression models in which the concentrations of the individual pollutants were replaced by their respective deciles. See statistical analysis in the methods section.

the highest for NO_2 and SO_2. However, a direct comparison of the magnitude of the RRs among the air pollutants can be misleading, because of differences in molecular weight. By contrast with our findings, a pooled analysis among European countries showed equivocal RRs of cardiovascular and respiratory deaths for NO_2 but significant RRs for black smoke, SO_2,and O_3.[29] The summary RRs for SO_2 and O_3 in western European cities are similar in magnitude to ours. In Hong Kong, the mean concentrations of O_3, NO_2, and PM_{10} are generally higher, but the concentrations of SO_2 are much lower than in European cities. Our findings of a significant RR of IHD for SO_2 suggest that the threshold has not been reached at the current SO_2 concentrations.

Owing to the high correlation between pollutants, it is not clear whether the observed effect of one pollutant in the single pollutant model represents that of an underlying pollutant. Much research focus has been given to the respirable fraction of particulates, and more recently, fine particulates ($PM_{2.5}$).[31 32] In the multipollutant model for both respiratory diseases and IHD, no significant effect for PM_{10} could be discerned. It should be noted that in some studies in the United States and Europe where the effects of particulates were significant, single pollutant models were used.[2–6 12–15 17 29] These results agreed with that of our single pollutant model for PM_{10}. Our findings of significant associations of SO_2 and O_3 with respiratory deaths are in accord with those in many European cities and elsewhere.[4 5 8 12 15 17 33–36] The problems of collinearity among pollutants in a multipollutant model are well recognised. In our data, the concentrations of NO_2 and PM_{10} were highly correlated on the same days (table 2), and on the best lag days that were used in the multipollutant model (r=0.73). The correlation between all the other pairs of pollutants ranged from trivial to moderate (Pearson's r ranged from –0.07 to 0.45 for the best lag days). Hence, it is statistically valid to include SO_2, O_3, and either of NO_2 and PM_{10} in the models. For all respiratory mortalities and COPD, the RR for O_3 was remarkably stable in all the models. A significant association with O_3 for respiratory mortality in our multipollutant model is biologically plausible. Firstly, O_3 is a highly toxic oxidant pollutant with known adverse effects on the respiratory system.[37] Secondly, the concentration of O_3 in Hong Kong is higher than in many cities in Europe and the United States, possibly related to the abundance of sunshine, even in winter months. In our earlier study on hospital admissions, O_3 was significantly associated with admissions for both respiratory and cardiovascular diseases.[21]

The high ambient concentration of NO_2 in Hong Kong is a possible explanation of our finding of a significant association with mortalities from IHD. Significant associations between NO_2 and all cardiovascular mortalities have been reported in some studies.[4 10 13] However, the relations between NO_2 and cardiovascular mortalities were inconsistent in a meta-analysis of pooled data from four European cities.[29] The reasons for the lack of association of PM_{10} with any respiratory or cardiovascular mortalities in a multipollutant model are not clear. The effects of PM_{10} might have been masked by those of NO_2 and O_3—the concentrations of both were fairly high. Neither could we explain why SO_2, rather than PM_{10}, was associated with deaths from pneumonia and influenza, especially when the mean concentration of SO_2 was relatively low and that of PM_{10} was fairly high. The chemical composition of particulates might be related to the effects on health. In Hong Kong, diesel vehicle exhaust is a major source of PM_{10}, although crustal dust and marine aerosols are important sources as well.[20]

No interaction was detected in this study between any two pollutants, whether analysed in pairs or when all four pollutants were considered together. Significant interactions between particulates and SO_2 have been reported in Athens and Lyon in the APHEA mortality studies.[15 33] The discrepancies might be due to differences in concentrations of pollutant between Hong Kong and these cities. The absence of interaction with the cold season could be related to the relatively mild winters in Hong Kong, where the mean monthly temperatures from December to February ranged from 15° to 19°C and the mean daily temperature rarely drops below 10°C. When analyzed by deciles of pollutant concentrations, a dose-response relation was found for all pollutants and respiratory mortalities, with no evidence of a threshold effect.

The role of CO on cardiovascular mortalities has been investigated elsewhere. Touloumi *et al* reported a positive association between ambient CO concentrations and daily mortality in Athens,[38] but this association was reduced considerably after including CO and black smoke or SO_2 in the same model. Carbon monoxide was not considered as a copollutant in other time series analyses.[39] Owing to the insufficiency of data, we did not evaluate the effect of CO, which might be relevant to cardiovascular mortalities. The ambient concentration of CO in Hong Kong is low. The mean 24 hour concentration was 800 $\mu g/m^3$, less than one tenth of the local air quality objective of 10 000 $\mu g/m^3$.[20]

The absence of a harvesting effect of respiratory and cardiovascular mortalities within a 3 week period suggests that the shortening of life is likely to be substantial. Schwartz suggested that deaths from chronic obstructive airway diseases were mostly brought forward by a few months, whereas some (but not most) deaths from pneumonia might be brought forward by a few days.[40] We have not investigated the harvesting effect of specific causes of death on different time scales.

In conclusion, we found significant associations of all four air pollutants with mortalities from respiratory disease and IHD, the effect size being strongest for COPD. Although the multipollutant model selected SO_2 and O_3 as significant pollutants for respiratory deaths and NO_2 for deaths from ischaemic heart disease, other pollutants such as PM_{10} might play an important part. The joint effects of the air pollutants are still poorly understood and comparative studies in cities with different pollutant profiles are warranted. The ecological design of the study precludes the inference of cause and effect.

Key messages

- Significantly increased risks of mortality from all respiratory diseases and ischaemic heart diseases were shown at current ambient concentrations of particulates, sulphur dioxide, nitrogen dioxide, and ozone.
- The associations between mortality and particulates and some gaseous pollutants concurred with many studies in temperate countries.
- The associations were not explained by "harvesting" of vulnerable populations and the effect of air pollution was likely to be real.
- A dose-response relation between the relative risks and the concentrations of the pollutants was found for all four air pollutants.
- When all pollutants were included in the models, ozone and sulphur dioxide remained significantly associated with respiratory mortalities, whereas nitrogen dioxide was associated with mortality from ischaemic heart disease.

Policy implications

- Current air quality standards are not sufficient to protect the public from adverse health effects.
- Further control of air pollution is likely to result in health benefits.

As the current ambient concentrations of these pollutants seem to be above the threshold, some health benefits should be noticeable if air pollution is reduced. A reduction in morbidity and mortality after the implementation of an intervention programme will add evidence to the hypothesis of a causal link between air pollution and ill health.

Authors' affiliations
T W Wong, W S Tam, T S Yu, A H S Wong, Department of Community and Family Medicine, The Chinese University of Hong Kong, 4/F School of Public Health, Prince of Wales Hospital, Shatin, N T, Hong Kong

REFERENCES

1 **Schwartz J**. Particulate air pollution and daily mortality in Detroit. *Environ Res* 1991;**56**:204–13.
2 **Schwartz J**, Dockery D. Particulate air pollution and daily mortality in Steubenville, Ohio. *Am J Epidemiol* 1992;**135**:12–19.
3 **Schwartz J**. Air pollution and daily mortality in Birmingham, Alabama. *Am J Epidemiol* 1993;**137**:1136–47.
4 **Anderson R**, Ponce de Leon A, Bland J, *et al*. Air pollution and daily mortality in London, 1987–92. *BMJ* 1996;**312**:665–9.
5 **Kelsall JE**, Samet JM, Zeger SL, *et al*. Air pollution and mortality in Philadelphia, 1974–88. *Am J Epidemiol* 1997;**146**:750–62.
6 **Wordley J**, Walters S, Ayres JG. Short term variations in hospital admissions and mortality and particulate air pollution. *Occup Environ Med* 1997;**54**:108–16.
7 **Hong YC**, Leem JH, Ha EH, *et al*. PM_{10} exposure, gaseous pollutants, and daily mortality in Inchon, South Korea. *Environ Health Perspect* 1999;**107**:873–8.
8 **Morgan G**, Corbett S, Wlodarczyk J, *et al*. Air pollution and daily mortality in Sydney, Australia, 1989 through 1993. *Am J Public Health* 1998;**88**:759–64.
9 **Peters A**, Skorkovsky J, Kotesovec F, et al. Associations between mortality and air pollution in Central Europe. *Environ Health Perspect* 2000;**108**:283–87.
10 **Mar TF**, Norris GA, Loenig JQ, *et al*. Associations between air pollution and mortality in Phoenix, 1995–1997. *Environ Health Perspect* 2000;**108**:347–53.
11 **Hales S**, Salmond G, Town GI, *et al*. Daily mortality in relation to weather and air pollution in Christchurch, New Zealand. *Aust N Z J Public Health* 1999;**24**:89–91.
12 **Simpson RW**, Williams G, Petroeschevsky A, *et al*. Associations between outdoor air pollution and daily mortality in Brisbane, Australia. *Arch Environ Health* 1997;**52**:442–54.
13 **Bremmer SA**, Anderson HR, Atkinson RW, *et al*. Short term association between outdoor air pollution and mortality in London 1992–4. *Occup Environ Med* 1999;**56**:237–44.
14 **Gouvea N**, Fletcher T. Time series analysis of air pollution and mortality: effects by cause, age and socioeconomic status. *J Epidemiol Community Health* 2000;**54**:750–5.
15 **Touloumi G**, Samoli E, Katsouyanni K. Daily mortality and "winter type" air pollution in Athens, Greece: a time series analysis within the APHEA project. *J Epidemiol Community Health* 1996;**50**(suppl 1):S47–51.
16 **Kunst AE**, Looman CWN, Mackenbach JP. Outdoor air temperature and mortality in the Netherlands: a time-series analysis. *Am J Epidemiol* 1993;**137**:331–41.
17 **Sunyer J**, Castellsague J, Saez M, *et al*. Air pollution and mortality in Barcelona. *J Epidemiol Community Health* 1996;**50**(suppl 1):S76–80.
18 **Xu X**, Dockery DW, Gao J, *et al*. Air pollution and daily mortality in residential areas of Beijing, China. *Arch Environ Health* 1994;**49**:216–22.
19 **Lee JT**, Shin D, Chung Y. Air pollution and daily mortality in Seoul and Ulsan, Korea. *Environ Health Perspect* 1999;**107**:149–54.
20 **Environmental Protection Department**. *Air quality in Hong Kong 1998*. Hong Kong: Hong Kong Government, 1999:3–13.
21 **Wong TW**, Lau TS, Yu TS, *et al*. Air pollution and hospital admissions for respiratory and cardiovascular diseases in Hong Kong. *Occup Environ Med* 1999;**56**:679–83.
22 **Katsouyanni K**, Schwartz J, Spix C, *et al*. Short term effects of air pollution on health: a European approach using epidemiologic time series data: the APHEA protocol. *J Epidemiol Community Health* 1996;**50**(suppl 1):S12–18.
23 **Schwartz J**, Spix C, Touloumi G, *et al*. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J Epidemiol Community Health* 1996;**50**(suppl 1):S3–11.
24 **McCullagh P**, Nelder JA. *Generalized linear models*. London: Chapman and Hall, 1989:323–32.
25 **SAS Institute**. *SAS/STAT Software: The GENMOD Procedure, Release 6.09*, Cary, NC: SAS Institute, 1993. (SAS Technical Report P-243.)
26 **Box GP**, Jenkins GM, Reinsel GC. *Time series analysis: forecasting and control*. Englewood Cliffs, NJ, Prentice-Hall International, 1994:21–45.
27 **Akaike H**. Factor analysis and AIC. *Psychometrika* 1987**. 52**:317–32.
28 **Spix C**, Heinrich J, Dockery D, *et al*. Air pollution and daily mortality in Erfurt, East Germany, 1980–9. *Environ Health Perspect* 1993;**101**:518–26
29 **Zmirou D**, Schwartz J, Saez M, *et al*. Time-series analysis of air pollution and cause-specific mortality. *Epidemiology* 1998;**9**:495–503.
30 **Pope CA III**, Bates DV, Raizenne ME. Health effects of particulate air pollution: time for reassessment? . *Environ Health Perspect* 1995;**103**:472–580.
31 **Borja-Aburto VH**, Castillejos M, Gold DR, *et al*. Mortality and ambient fine particles in southwest Mexico City, 1993–5. *Environ Health Perspect* 1998;**106**:849–55.
32 **Norris G**, YoungPong SN, Koenig JQ, *et al*. An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Perspect* 1999:**107**:489–93.
33 **Zmirou D**, Barumandzadeh T, Balducci F, *et al*. Short term effects of air pollution on mortality in the city of Lyon, France, 1985–90. *J Epidemiol Community Health* 1996;**50**:S30–5.
34 **Spix C**, Wichmann HE. Daily mortality and air pollutants: findings from Koln, Germany. *J Epidemiol Community Health* 1996;**50**:S52–8.
35 **Vigotti MA**, Rossi G, Bisanti L, *et al*. Short term effects of urban air pollution on respiratory health in Milan, Italy, 1980–9. *J Epidemiol Community Health* 1996;**50**:S71–5.
36 **Saldiva P**, Lichtenfels A, Paiva PS, *et al*. Association between air pollution and mortality due to respiratory diseases in children in Sao Paulo, Brazil: a preliminary report. *Environ Res* 1994;**65**:218–25.
37 **Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society**. Health effects of outdoor air pollution. *Am J Crit Care Med* 1996;**153**:3–50.
38 **Touloumi G**, Pocock SJ, Katsouyanni K, *et al*. Short-term effects of air pollution on daily mortality in Athens: a time series analysis. *Int J Epidemiol* 1994;23:957–67.
39 **Verhoeff AP**, Hoek G, Schwartz J, *et al*. Air pollution and daily mortality in Amsterdam. *Epidemiology* 1996;**7**:225–30.
40 **Schwartz J**. Harvesting and long term exposure effects in the relation between air pollution and mortality. *Am J Epidemiol* 2000;**151**:440–8.